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Oral Medicine

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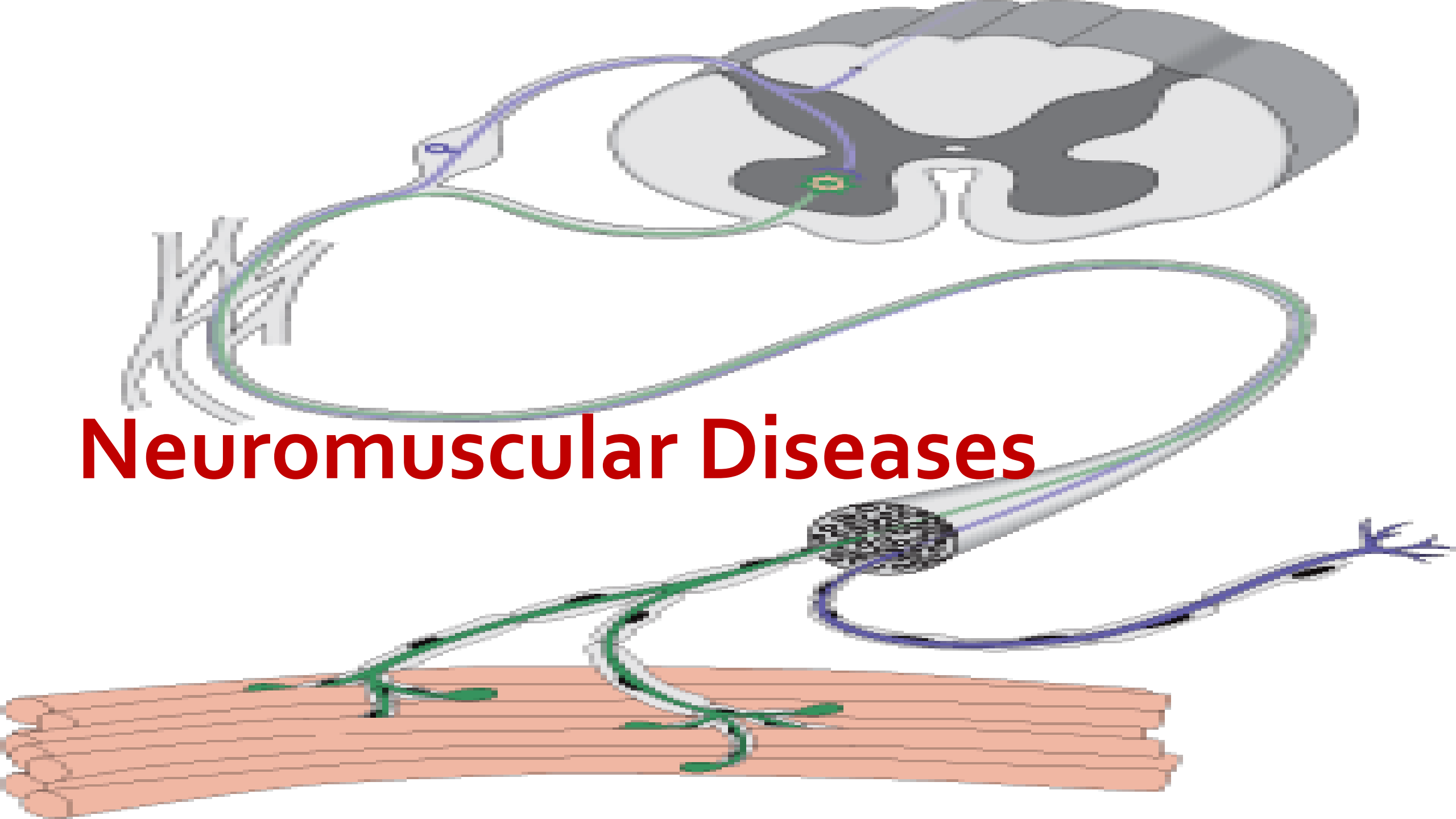


Neuromuscular Diseases

Cerebrovascular Disease

Cerebrovascular disease refers to disorders that result in damage to the cerebral blood vessels leading to impaired cerebral circulation.

Cerebrovascular accident (CVA), or complete stroke, is a sudden impairment in cerebral circulation resulting in death or a focal neurologic deficit lasting more than 24 hours.

Neurologic events related to CVA include:

- **Transient ischemic attack (TIA):** It's a reversible, acute, short-duration, focal neurologic deficit resulting from transient and localized cerebral ischemia (reversible within 24 hours)
- **Reversible ischemic neurologic defect (RIND):** It's a reversible, acute, focal neurologic deficit due to transient and localized cerebral ischemia but resulting in neurologic deficits that last more than 24 hours
- **stroke in evolution:** defined as progressive worsening of stroke symptoms.

Impaired cerebral blood flow leading to ischemia and energy failure is the common pathogenic mechanism for stroke.

A 50% decrease in blood flow to the brain for as few as 3 to 4 minutes can result in irreversible brain injury. Following infarction, edema and excessive neurotoxic excitation contribute to further regional tissue injury and death.

*15% of strokes result from hemorrhagic events leading to infarction, most often related to hypertension, trauma, substance abuse.

*85% of strokes result from ischemia due to atherosclerotic disease, thromboembolic events, and occlusion of cerebral blood vessels.

Diagnosis: 1.Thorough neurologic and cardiovascular examination.

2. Anatomic and functional brain imaging; Cerebral angiography and brain MRI are the most effective techniques for localizing the stroke site.

3. Laboratory evaluation of the stroke patient includes complete blood count, comprehensive metabolic panel, urinalysis, coagulation profile.

Treatment: The outcome of stroke and related TIAs and RIND is significantly affected by the timeliness of treatment.
Early intervention is critical to prevention, treatment, and recovery.

Management of acute stroke includes:

- medical therapy to reduce bleeding or thromboembolic occlusion.
- medical therapy to reduce brain edema and neurotoxicity/nerve injury.
- surgical interventions (revascularization, hemorrhage control).

Oral Health Considerations: Following stroke, patients may experience several oral problems, including
-masticatory and facial muscle paralysis,
-impaired or lost touch and taste sensation,
-diminished protective gag reflex,
and dysphagia.

Diminished motor function of masticatory and facial muscles will reduce food clearance from the mouth and teeth may adversely affect oral hygiene and increase the risk for caries and periodontal disease.

Creative and effective use of adjuvant oral hygiene techniques and devices (oral antimicrobial rinse, oral irrigation, floss holders) represents an important approach to oral health promotion and disease prevention, supported by frequent recall examination and prophylaxis.

Replacement of missing teeth and adequacy of removable and fixed prostheses are essential to effective chewing and diet.

Prior history of TIA or stroke increases the risk of a future or second stroke, with the highest risk during the first 90 days, leading to the recommendation that elective dental treatment be deferred for 6 months following a stroke or for a patient with active TIAs or RIND.

Use of antiplatelet and anticoagulant medications is common in patients with a history of stroke, TIA, and RIND, therefore bleeding time test is often recommended to evaluate the qualitative defect in platelets.

Concomitant use of nonsteroidal anti-inflammatory drugs (NSAIDs) may increase the risk for bleeding, and their long term use may reduce the protective effect of aspirin.

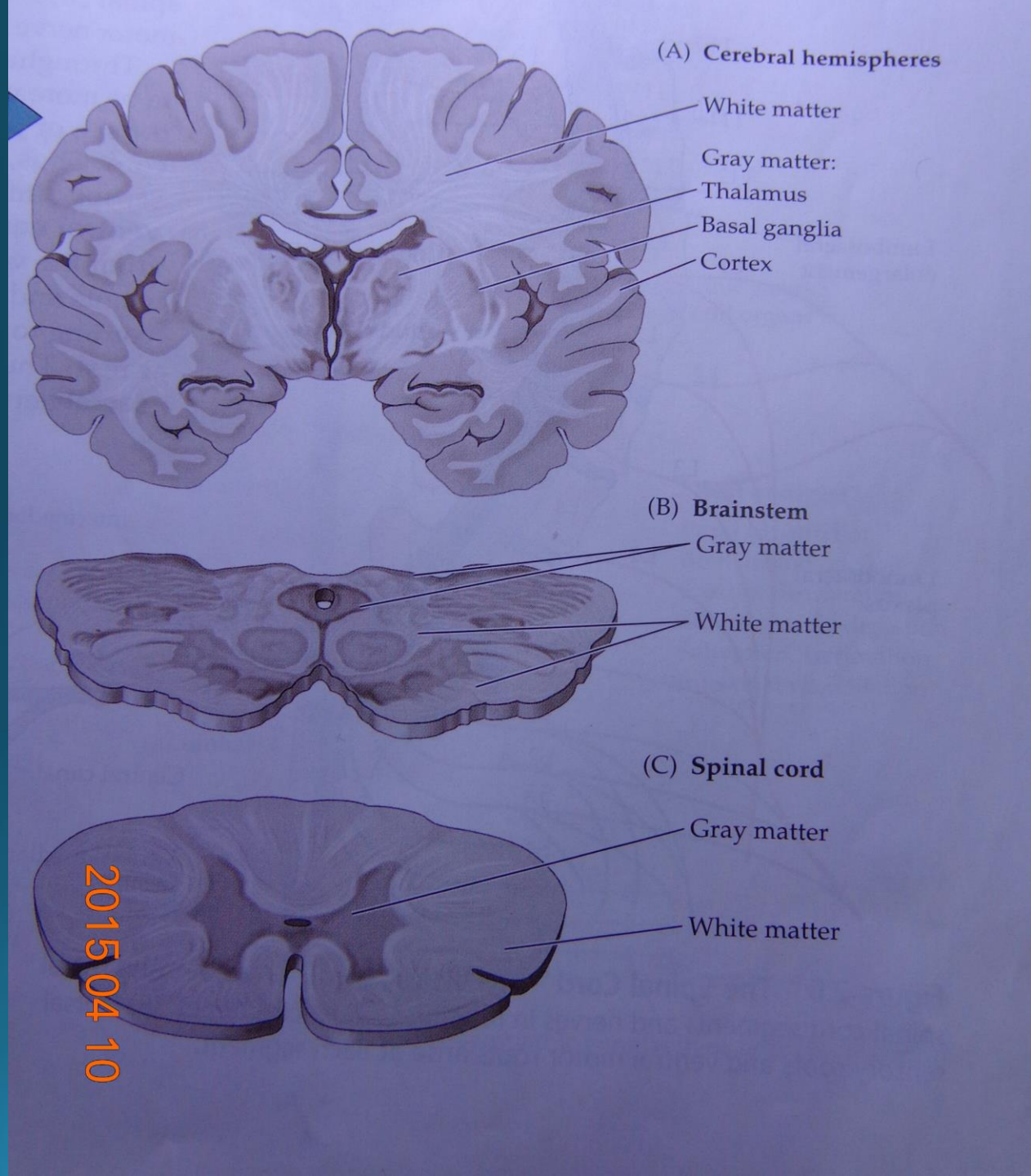
Stress reduction for the patient during dental visits is necessary to make the patient comfortable and minimize anxiety-related elevation in blood pressure.
Pre- or Perioperative inhalation N_2O-O_2 or oral anxiolytic medication can aid in reducing treatment-related stress and anxiety.

Use of epinephrine-containing local anesthetics is not strictly contraindicated, but they should be used minimally and generally follow guidelines recommended for patients with cardiovascular disease.

Blood pressure should be monitored at every visit and within a visit if long and stressful.

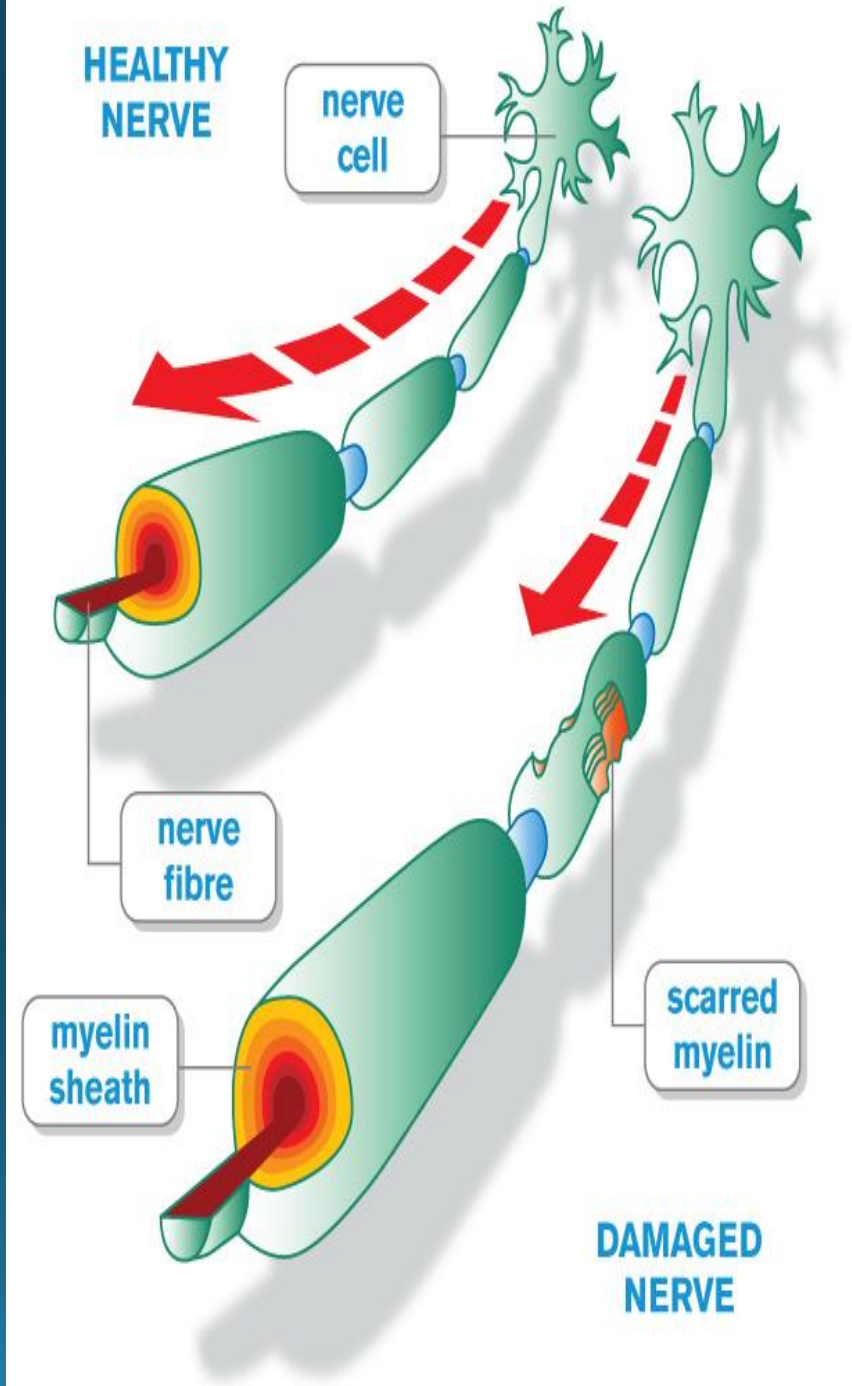
Multiple Sclerosis

MS is characterized by multiple areas of central nervous system (CNS) white matter inflammation, demyelination, and gliosis (scarring).



Myelin is critical for propagation of nerve impulses, and when it is destroyed in MS, slowing and/or complete block of impulse propagation are manifested by abnormal muscular and neurologic signs and symptoms.

The age at onset is typically between 20 and 40 years. MS is more common among women than men (2:1)ratio.

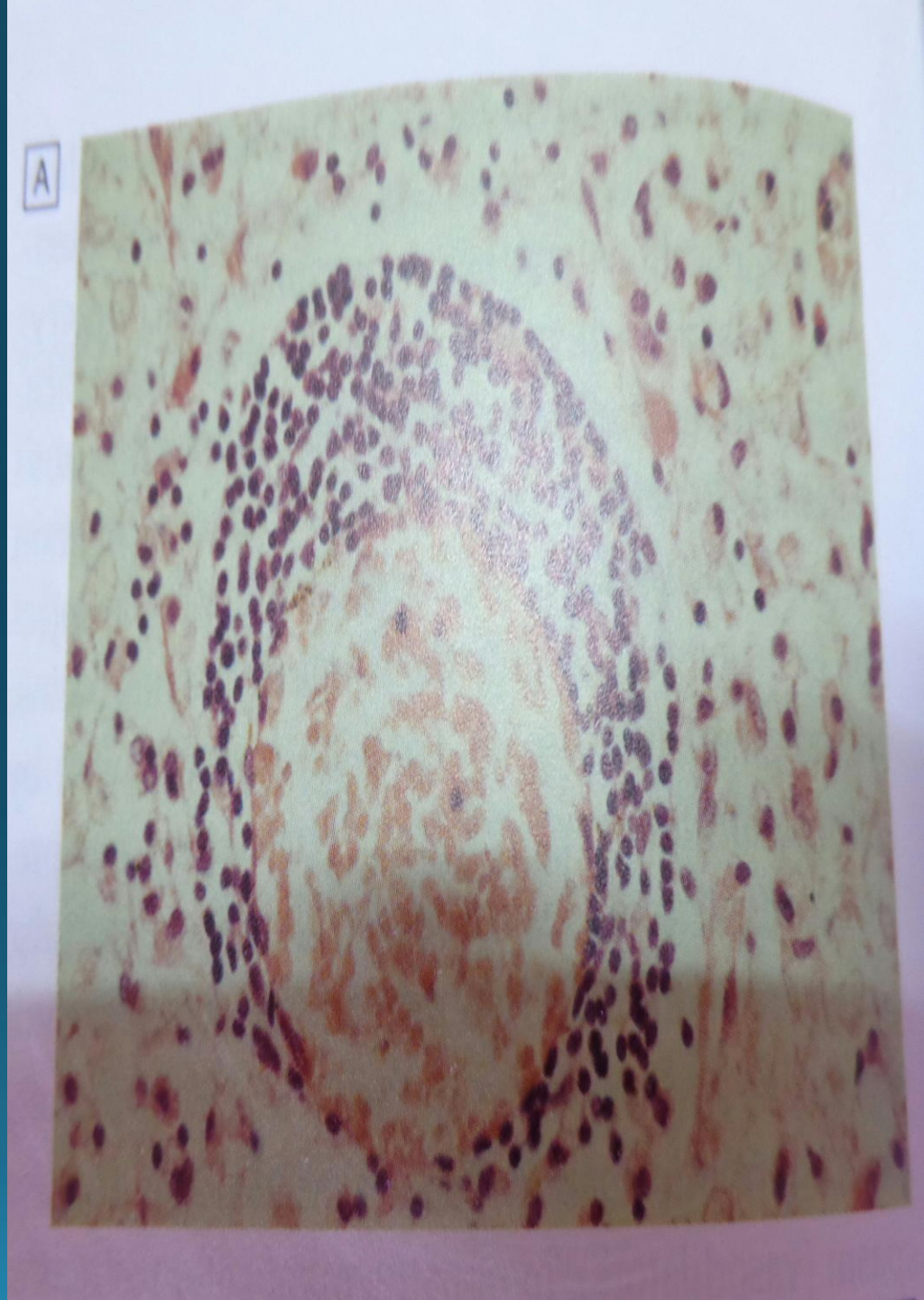


Although the cause of MS is unknown, genetic susceptibility to MS clearly exists, and it is thought that an initial trigger leads to autoimmune mechanisms causing demyelination.

Epidemiologic evidence supports the role of an environmental exposure in MS, and the two most common infectious agents to be implicated in the pathogenesis of this disease are Epstein-Barr virus and human herpesvirus 6.

MS lesions or "plaques" vary in size

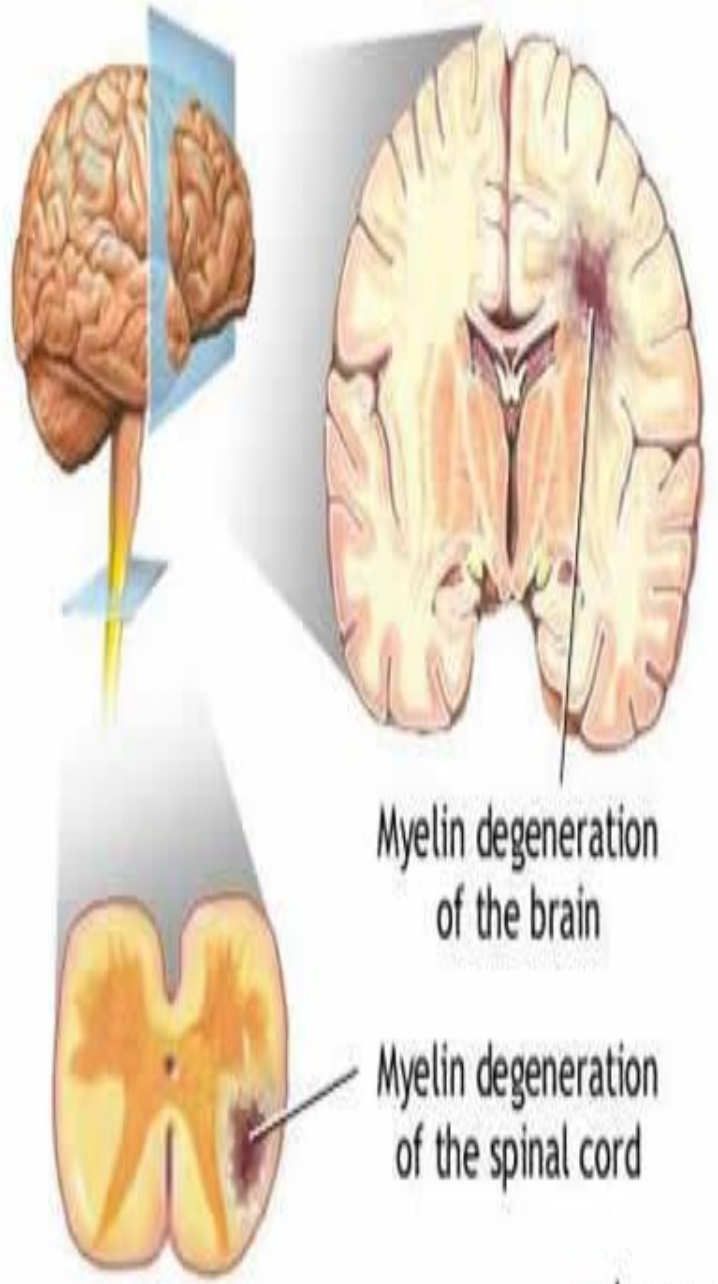
characterized by perivenular cuffing with inflammatory mononuclear cells, predominantly macrophages and T cells, that is generally limited to the white matter and periventricular areas of the CNS.



Plaques may be found in both the brain and spinal cord.

within the plaques, there is variable destruction of myelin and neuronal axons with preservation of the ground structure.

Uniform areas of incomplete myelination are called shadow plaques and may be evident in chronic lesions of MS.



The clinical manifestations of MS depend on the areas of the CNS involved, and frequently affected areas include the optic chiasm, brainstem, cerebellum, and spinal cord.

-The sudden onset of optic neuritis (diminished visual acuity, dimness, or decreased color perception),without any other CNS signs or symptoms, is often considered the first symptom of MS. Other common visual signs in patients with MS include diplopia, blurring, nystagmus, gaze disturbances and visual field defects.

- Limb weakness is characteristic of MS and can manifest as loss of strength or dexterity, fatigue, or gait disturbances.

- Spasticity associated with painful muscle spasms is often observed in the legs of patients with MS and may interfere with a patient's ability to ambulate.

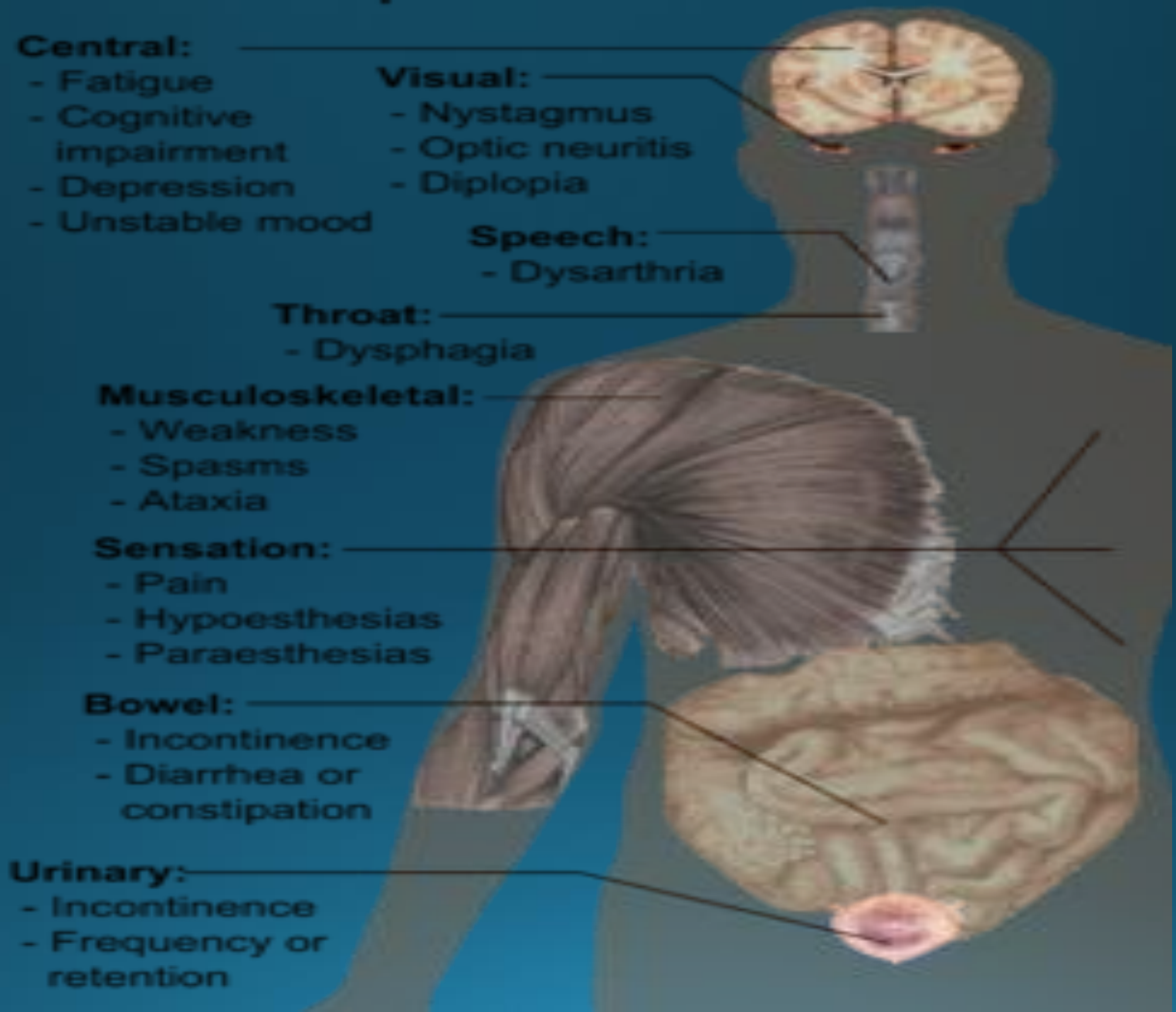
-Ataxia may affect the head and neck of MS patients and may result in cerebellar dysarthria (scanning speech).

- Bladder dysfunction and bowel dysfunction frequently coexist and are present in >90% of MS patients.

- MS patients often demonstrate sensory impairment, including paresthesia and hyperesthesia. Fatigue, depression, and cognitive dysfunction are often observed in patients with MS.

Main symptoms of

Multiple sclerosis



Diagnosis: There is no definitive diagnostic test for detection of MS.

Clinical criteria for MS include two or more episodes of symptoms and two or more signs of pathology affecting white matter tracts of the CNS.

MRI demonstrates characteristic abnormalities of MS in >95% of patients.

CSF is often analyzed in patients suspected of having MS, and positive findings include an *increase in total protein and mononuclear white blood cells*. In addition, there is often an *increase in synthesized IgG* in patients with MS.

Treatment: Therapy for MS can be divided into three categories:

(1) treatment of acute attacks.

(2) treatment that reduces the biologic activity of MS.

(3) symptomatic therapy.

Glucocorticoids are used to manage both initial attacks and acute exacerbations of MS. Intravenous methylprednisolone is administered at a dose between 500 and 1,000 mg/d for 3 to 5 days to reduce the severity and length of an attack.

Agents that reduce the biologic activity of MS include interferon (IFN)-b1a.
IFN-b1b.
glatiramer acetate.
All four pharmacologic agents slow progression of relapsing disease and reduce the annual relapse rate by 20 to 40%.
Common agents employed for management of MS symptoms include potassium channel blockers for weakness, lioresal for spasticity, propantheline for bladder dysfunction, and amantadine for fatigue.

Oral Health Considerations: Individuals may present to the oral health care provider with signs and symptoms of MS.
-Trigeminal neuralgia (TGN), which is characterized by electric shock–like pain, may be an initial manifestation of MS in 0.3% of cases. Features of MS-related TGN include possible absence of trigger zones and continuous pain with lower intensity.

-Patients with MS may also demonstrate neuropathy of the (V2) and (V3) branches of the trigeminal nerve, which may include burning, tingling, and/or reduced sensation. Neuropathy of the mental nerve can cause numbness of the lower lip and chin.

-Myokymia may be seen in patients with MS secondary to MS lesions affecting the facial nerve.

- Facial weakness and paralysis may also be evident in MS patients.

- Dysarthria that results in a scanning speech pattern is often seen in patients with MS.

-If MS is suspected, oral health care professionals should carefully evaluate cranial nerve function. If cranial nerve abnormalities are detected upon examination, the individual should be referred to a neurologist for further evaluation.

-It is recommended to avoid elective dental treatment in MS patients during acute exacerbations of the disease due to limited mobility and possible airway compromise.

- Patients with significant dysfunction may require dental treatment in an operating room under general anesthesia due to the inability to tolerate treatment in an outpatient setting.

Seizure Disorders

A seizure is a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from neuronal aggregates in the CNS.

epilepsy is a condition in which aperson has recurrent seizures due to a chronic, underlying process.

classification system of the epilepsies and epileptic syndromes based on the clinical features of seizure activity and associated EEG changes.

The major categories of seizure activity used in clinical practice include:

- Partial seizures
- Generalized seizures

* Absence seizures (petit mal)
* Tonic-clonic (grand mal)
*atypical absence seizures .
*atonic seizures .
*myoclonic seizures.

- unclassified seizures

Clinical Manifestations

Partial Seizures

Simple partial seizures are not associated with impaired consciousness.

Simple partial seizures consist of clonic activity, which are rapid jerks that also can be accompanied by somatosensory phenomena, visual changes/distortions, and auditory, olfactory, and gustatory symptoms.

Complex partial seizures result in either a loss or impairment of consciousness.

Many of these seizure foci originate within the temporal and inferior frontal lobes, causing patients to appear confused and to experience visual or auditory hallucinations.

The seizures frequently begin with an aura (a warning of impending seizure activity) that may consist of a sense of fear, detachment, and/or intense odors/sounds.

Generalized Seizures

*Absence seizures are characterized by seconds of unconsciousness with no loss of body tone. In addition, subtle facial twitching and rapid eye blinking are often observed without generalized clinical muscular activity.

* Tonic-clonic seizures characteristically begin abruptly and may or may not be preceded by an aura. The patient loses consciousness, while the entire musculature contracts forcibly, lasting between 20 and 40 seconds. Contraction of the muscles of the larynx and forced expiration can produce a loud moan, often termed "epileptic cry".

Patients gradually regain consciousness and often complain of fatigue and headache after a tonic-clonic seizure.

Diagnosis:

- History and physical examination are critical as the diagnosis of a seizure may be based on clinical findings only. A complete neurologic examination is required for all patients with suspected seizure activity, including testing of cranial nerve function, assessment of mental status, and testing of motor function.
- Blood studies, such as a complete blood count, electrolytes, glucose, magnesium, and calcium, are performed routinely to identify metabolic causes of seizure activity.
- screening tests include toxin screens to identify seizure activity due to drugs.
- lumbar puncture to rule out any infectious etiologies

- MRI is the diagnostic modality of choice for the detection of malformations of cortical development, vascular malformations, tumors, and acquired cortical damage, all of which are common etiologies for seizure disorders.

- CT is valuable for investigating intracranial calcification, skull fractures, and suspected CNS infection.
- An EEG is mandatory for patients with suspected seizures and is critical for classifying seizure disorders as well as helping to determine the type of anticonvulsant medication, if indicated.

Treatment:

For patients with recurrent seizures without identifiable causative pathology, *pharmacologic therapy is initiated. The goal of pharmacologic therapy is to choose a medication that is most appropriate for the specific type of seizure activity and to administer it in the proper dose to achieve control of seizure activity with minimal side effects.

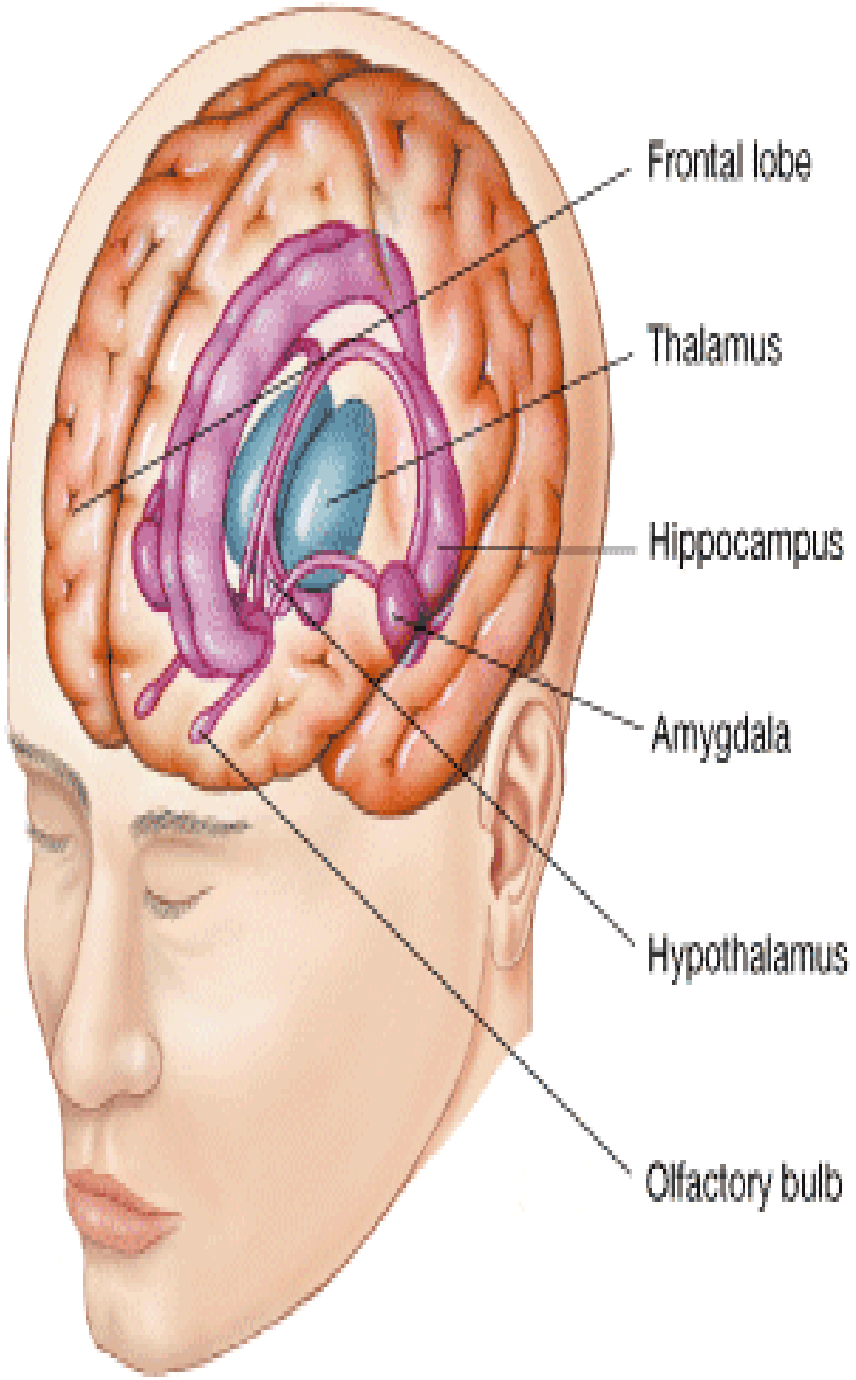
-Lamotrigine, carbamazepine, or phenytoin is currently the initial drug of choice for the treatment of partial seizures.
Phenytoin is associated with gingival overgrowth, hirsutism, and coarsening of facial features. Carbamazepine can cause hepatotoxicity, leukopenia, and aplastic anemia, whereas lamotrigine has been associated with skin rash.

- Valproic acid is the best initial choice for treatment of generalized tonic-clonic seizures.It may cause bone marrow suppression and hepatotoxicity, requires laboratory monitoring, and should be avoided in patients with preexisting bone marrow or liver disease

-Ethosuximide has been shown to be particularly effective for the treatment of uncomplicated absence seizures.

*vagus nerve stimulation, is indicated patients who are not candidates for resective brain surgery.

*Surgical procedures may be indicated for patients who are resistant to all medical therapies, including limited removal of the hippocampus and amygdala, temporal lobectomy, or hemisperectomy



Oral Health Considerations:

-A complete evaluation of a patient's seizure disorder is necessary prior to initiation of any dental treatment to determine the stability of the condition and an appropriate venue for treatment.

Important features for the clinician to assess include the type of seizures, etiology of seizures, frequency of seizures, known triggers of seizure activity, presence of aura prior to seizure activity, and history of injuries related to seizure activity.

If a patient demonstrates signs of poorly or uncontrolled seizure disorder, consultation with the patient's physician and/or neurologist is recommended.

-Patients with poorly or uncontrolled seizure disorder may not be suited for private dental offices and should be referred to a hospital setting for routine dental care.

-Patients with poorly controlled disease or stress-induced seizures may require sedative medications prior to treatment; this should be determined in consultation with the patient's physician.

-Placement of metal fixed prostheses is recommended rather than removable prostheses to decrease the risk of displacement and aspiration risk during seizure activity.

-Phenytoin, carbamazepine, and valproic acid can cause bone marrow suppression, leukopenia, thrombocytopenia, and secondary platelet dysfunction, resulting in an increased incidence of microbial infection, delayed healing, and both gingival and postoperative bleeding. Patients taking these medications may require laboratory evaluation prior to dental treatment, including a complete blood count with differential to assess white blood cell and platelet counts and coagulation studies to assess clotting ability.

-Patients on long-term carbamazepine should have serum blood levels evaluated prior to initiating dental treatment as insufficient doses may result in inadequate seizure control and excessive doses have been associated with hepatotoxicity.

-Aspirin and nonsteroidal anti-inflammatory medications should be avoided for postoperative pain control in patients taking valproic acid as they can enhance the possibility of increased bleeding.

-Gingival overgrowth is a significant oral complication among seizure disorder patients taking anticonvulsant medications, most notably phenytoin.
Inflammation can exacerbate this condition; therefore, frequent professional cleanings and use of an electric toothbrush are recommended to maintain optimal oral hygiene. Use of chlorhexidine and/or folic acid rinses to minimize gingival inflammation among seizure disorder patients with gingival overgrowth.

-Xerostomia may result from the use of antiseizure medications, and oral health care providers may observe increased dental caries and oral candidiasis in patients using these agents. Topical fluoride should be considered for patients with seizure disorders who are at increased risk of developing dental caries, and antifungal agents should be prescribed if oral candidiasis develops.

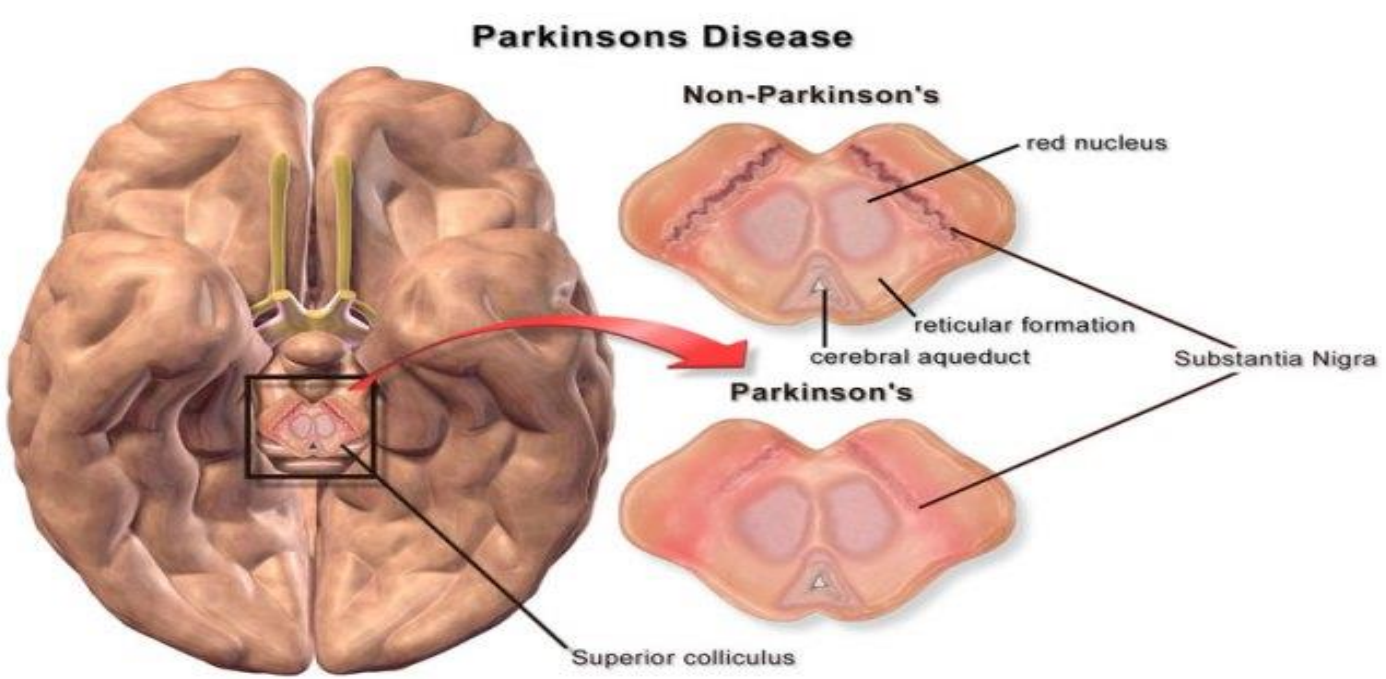
Parkinson's Disease(PD):

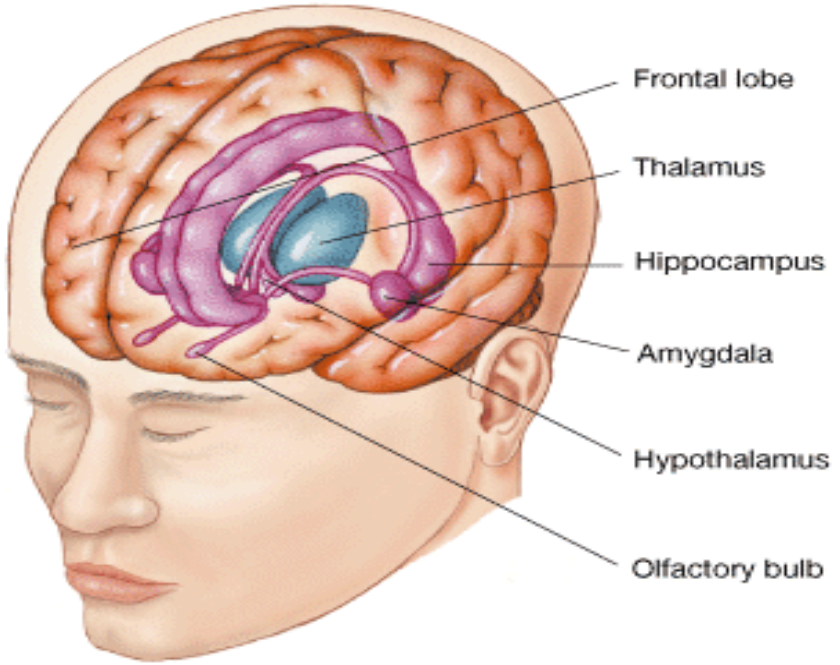
It is a chronic, progressive, neurodegenerative disorder characterized by resting tremor, rigidity (feeling of periodic resistance to passive movement owing to co-contraction of agonist and antagonist muscle pairs), and bradykinesia (slow intentional movements).

Prevalence and incidence increase with age.

There may be a small increased risk for PD among men compared with women, and all races and ethnic groups are affected equally.

PD results from idiopathic degeneration of the dopaminergic cells in the pars compacta of the substantia nigra, leading to depletion of the neurotransmitter dopamine in the basal ganglia (caudate nucleus and putamen).





Clinical Manifestations: The four cardinal signs of PD are
- resting tremor (in hands, arms, legs, jaw, and face)
- rigidity or stiffness (limbs and trunk)
- bradykinesia (slowness of movement)
- postural instability or impaired balance and coordination.

Secondary symptoms include change in speech, difficulty in swallowing, pain, confusion, depression, fatigue, and constipation.

Diagnosis: The diagnosis is usually based on the health history and a neurologic examination;

when symptoms are subtle and the presentation is incomplete, the diagnosis can be difficult. Anatomic and functional brain imaging, CSF evaluation, and laboratory testing are often necessary to exclude other diagnoses.

Treatment: There is no cure for PD, but a variety of medications and procedures provide relief from the symptoms.

-The most common pharmacologic treatment is dopamine replacement therapy using levodopa (used by neurons to synthesize dopamine) combined with carbidopa (delays the conversion of levodopa into dopamine until it reaches the brain).

-Surgical management of PD is more often selected in younger patients with advanced PD or intolerable medication side effects.

-Levodopa and dopamine agonists can lead to:
*orthostatic hypertension and, rarely, severe hypertension therefore monitoring of blood pressure correct positioning and repositioning during and after treatment.
* oromandibular and facial dyskinesia so impact of oromandibular dyskinesia on the design of dental prostheses.
*xerostomia xerostomia and caries risk reduction through hygiene, sealants and fluorides when indicated.
*blood dyscrasias therefore periodic evaluation of the complete blood count to detect drug-related hematologic adverse effects.

Oral Health Considerations:

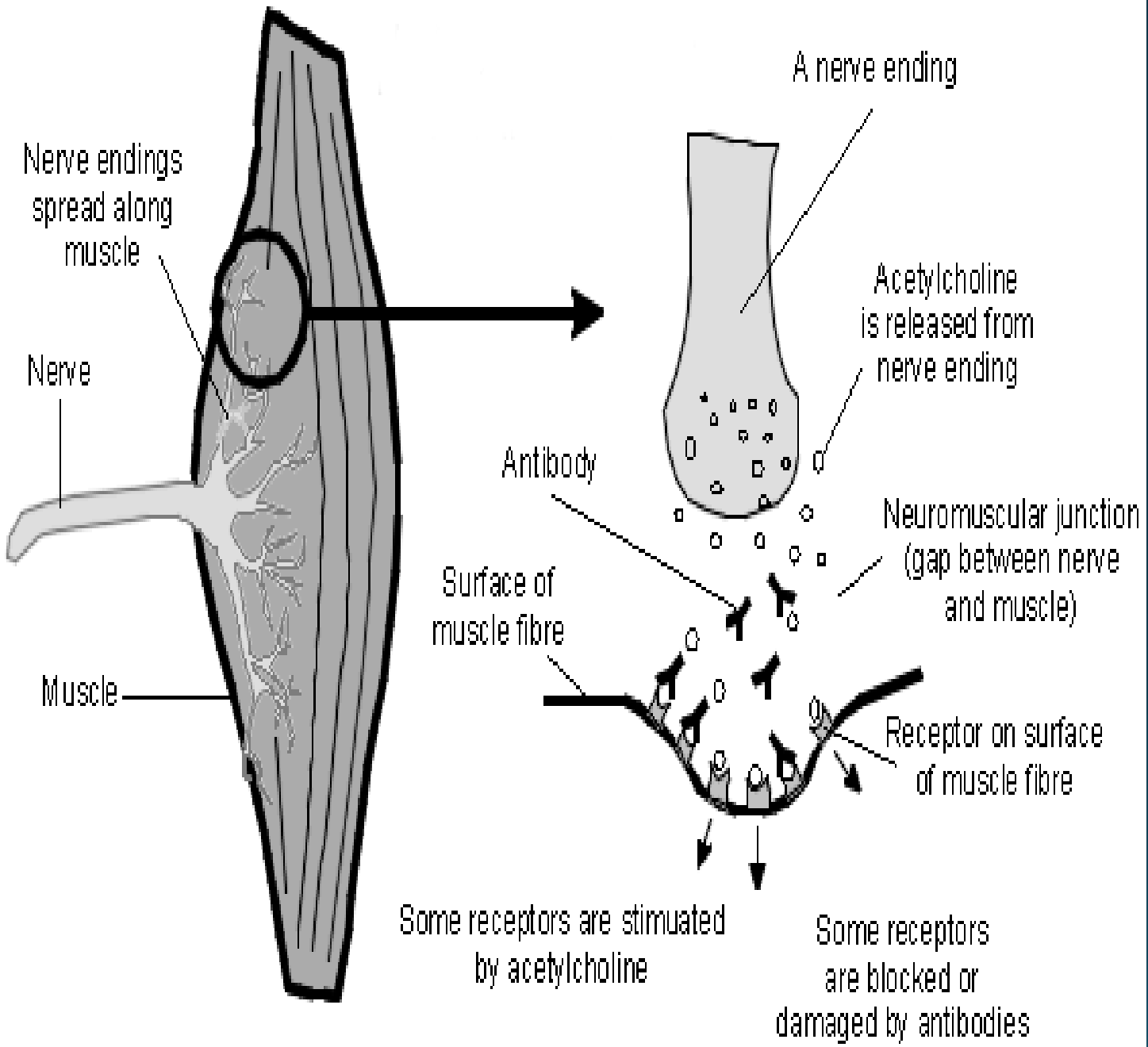
-Patients with PD often must be treated in a relatively upright position.

-Resting tremors and drug-related dyskinesia can complicate procedures, and behavioral techniques to reduce anxiety as well as gentle cradling techniques can help.

-Dysphagia and impaired gag reflex increase the risk for aspiration of oral and irrigation fluids, and high-speed evacuation of fluids is important in reducing the risk for aspiration pneumonia.

Myasthenia Gravis (MG)

It is a chronic neuromuscular disease caused by autoimmune destruction of the acetylcholine receptor (AChR), disrupting cholinergic neuromuscular neurotransmission.



Women are more commonly affected than men, with an onset in the second and third decades.

10% of patients with MG have a thymic tumor and 70% have hyperplastic changes that indicate an active immune response. Removal of the tumor can improve symptoms dramatically.

Clinical Manifestations:

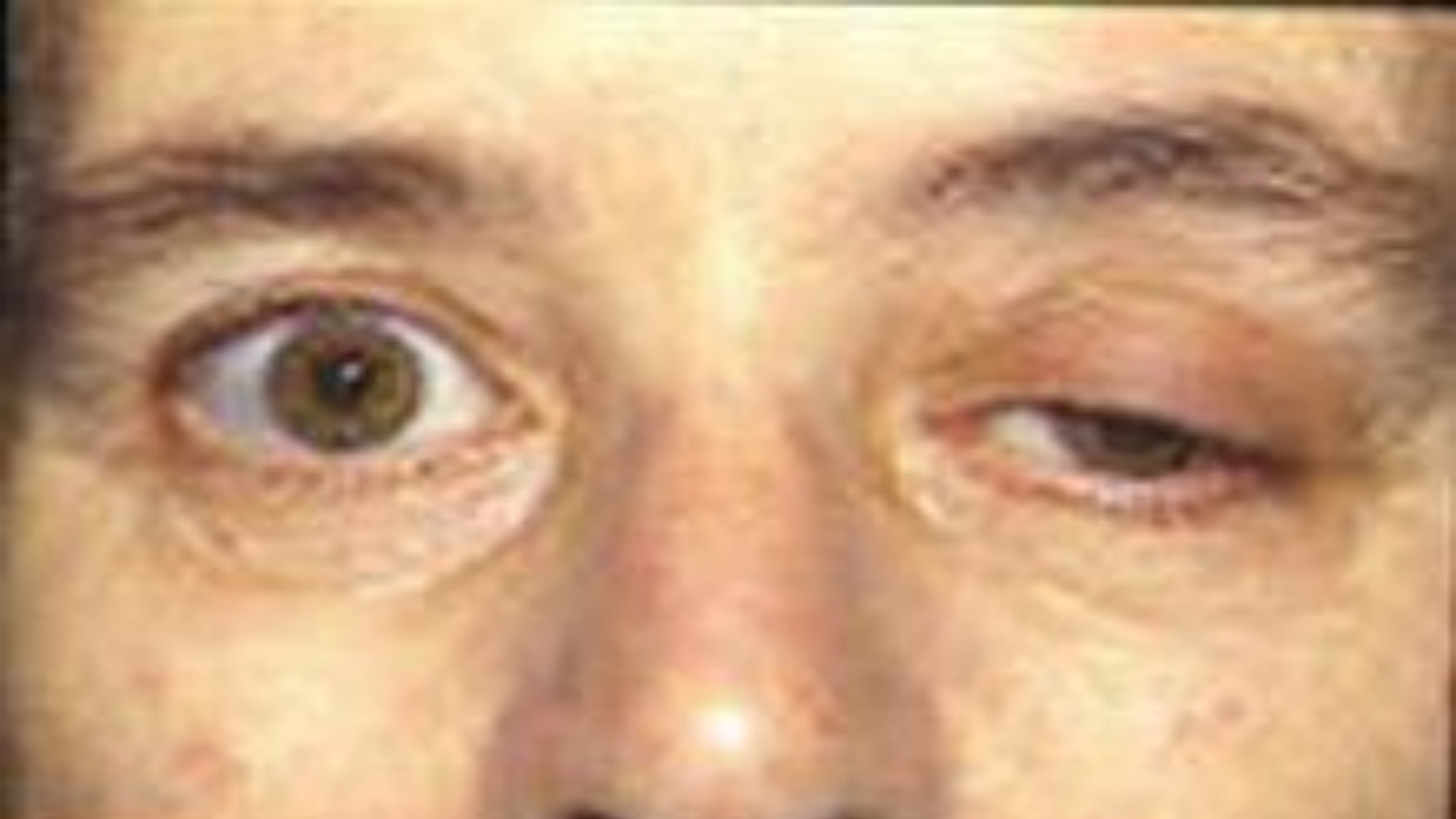
-Two-thirds of patients with MG present with a complaint of specific muscle weakness in the eyes (with associated diplopia) and/or drooping eyelids.

-Oropharyngeal muscle weakness,difficulty in chewing, swallowing, or talking, is the initial symptom in one-sixth of patients.

-limb weakness in only 10%.

The severity of weakness fluctuates during the day, usually best in the morning and worse as the day progresses.

Factors that worsen MG symptoms include stress, systemic illness (especially viral respiratory infections), hypothyroidism or hyperthyroidism, pregnancy, and menses.



Diagnosis:

*clinical examination and history.
*serologic evidence of autoantibodies to the AChR.
*CT or MRI of the chest is highly accurate in detecting thymoma, and every patient with MG should be screened for thymoma.

Treatment

-Anticholinesterase drugs such as neostigmine and pyridostigmine bromide increase acetylcholine availability and receptor binding and provide symptomatic benefit without influencing the course of the disease.

-Patients with thymus tumors may have dramatic improvement following thymectomy.

-Patients with more severe symptoms or poor response to treatment have treatment directed at reducing autoantibody production using corticosteroids (oral or intravenous pulse therapy).

-plasmapheresis to remove autoantibodies.

- high-dose intravenous immunoglobulin.

Oral Health Considerations

Orofacial signs and symptoms are an important presenting feature of MG, and the dental provider may be in the position to recognize and refer for diagnosis.

-difficulty in chewing can affect diet and the design of prostheses.

-Aspiration risks can be high and can be reduced by adequate suction and the use of a rubber dam.

-The MG patient may also be at risk for a respiratory crisis from the disease itself or from overmedication; if this is a substantial risk and the dental treatment is necessary, dental treatment in a hospital should be considered where endotracheal intubation can be performed.
-Avoid prescribing drugs that may affect the neuromuscular junction, such as narcotics, tranquilizers, and barbiturates.
-Certain antibiotics, including tetracycline, streptomycin, sulfonamides, and clindamycin, can affect neuromuscular activity and should be avoided or used with caution.